Simulation of the Date of Maturity of *Plasmopara viticola* Oospores to Predict the Severity of Primary Infections in Grapevine

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ABSTRACT

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A study of the dynamics of *Plasmopara viticola* oospore maturation during 3 yr in the Bordeaux area of France showed significant differences among years. The date of oospore maturity and subsequent disease severity in spring appeared to be associated with the amount of rainfall after oospore formation. For example, heavy rainfall from September to February was generally associated with early oospore maturity and severe disease in May or June. To predict the date when most oospores are mature (DOM), a climate-based model called "Prediction of Oospore Maturity" (POM) was developed. The POM model, based on daily rainfall beginning in September, permits calculation of DOM as early as the end of January. POM calculations based on climatic data recorded since 1977 confirmed that the earlier the DOM, the more severe disease was in the spring. Four levels of predictable risk are proposed based on the time required for oospore maturation. Although the POM model needs validation, it already shows promise in grape disease management.

Additional keywords: downy mildew, epidemiology, risk modeling

Oospores constitute the primary means of winter survival of *Plasmopara viticola* (Berk. & Curt.) Berl. & de Toni and are the initial inoculum for grapevine downy mildew. Previous studies of this organism have focused on the prediction of primary infection dates in spring and have not allowed precise determination of the relationship between the evolution of oospore maturation and the severity of subsequent infections. However, simulations of disease risk made with the "EPI model" developed by Strizyk (11,12) showed that winter climatic data must be incorporated to give accurate predictions (6,8). As early as the end of March, the model provides an indication of primary infection risk by using climatic data starting from October. According to simulations conducted for a 12-yr period (1977–1988), risk is highest when rain is abundant during the oospore maturation period.

Using a technique for assessing the dynamics of oospore maturation and germination (9), we tried to better understand the role of oospores in an epidemic. We studied the maturation of oospores and the initial development of downy mildew in the Bordeaux area of France over 3 yr and developed a model for predicting the dates of oospore maturity. The a posteriori validation of this model over several years demonstrates its ability to predict disease severity in spring (May–June).

MATERIALS AND METHODS

Dynamics of oospore maturation. Infected leaves containing oospores of *P. viticola* were collected on 24 October 1984, 22 October 1985, and 20 October 1987 from an experimental vineyard belonging to the INRA Research Center in Bordeaux. Under a binocular microscope, leaf disks 6 mm in diameter containing oospores (more than 1,000/cm^2) were punched out with a cork-borer and stored in plaster modeling tubes (40 mm long, inner diameter 12 mm, outer diameter 30 mm) (9). Tubes containing leaf disks were buried under 5 cm of soil and exposed to natural vineyard conditions. Disks were subsampled from the tubes every 15 days beginning in January. Two hundred oospores per sample were dissected from the leaf disks and placed on 1% water agar in petri dishes. The percentage of oospores capable of germinating at 20 C in a lighted incubator (i.e., the percentage of mature oospores) was assessed daily with a microscope (9).

Climatic records. Climatic conditions were recorded by the Regional Service of the National Meteorological Institute. They consisted of daily rainfall in millimeters (*Rd*) from 21 September 1977 to 31 March 1988 and monthly average rainfall (*RM*) calculated from 1946 to 1987.

Assessment of the intensity of primary infections. Disease severity of downy mildew was rated annually from 1977 to 1988 on a scale of 1–4 from the observations published by Plant Protection Services in forecasting bulletins (8). Disease ratings were based on the severity of early downy mildew on leaves and bunches (gray rot symptoms) until end of bloom. Ratings were made in the Bordeaux production area (100,000 ha) on susceptible cultivars. The disease ratings, followed by the years given each rating from 1977 to 1988, are as follows: 1 = disease absent or minimal, with no economic incidence before véraison (1984, 1986, 1987); 2 = disease present in a few vineyards, inducing moderate but not severe damage (1978, 1979, 1981); 3 = disease present in most vineyards in the area, sometimes very severe and leading to significant economic losses (1980, 1982, 1983); and 4 = disease present in almost all vineyards, inducing very severe damage (100% crop loss in vineyards not properly treated with fungicides) (1977, 1985, 1988).

RESULTS

Biological basis for the model. *Dynamics of oospore maturation in 1985, 1986, and 1988.* Our study of the development of mature oospores in 1985, 1986, and 1988 showed that the maturation period can be subdivided into three phases (Fig. 1). In the first phase,

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oospores are not able to germinate. In the second or maturation phase, the proportion of mature oospores rises until an optimum is reached where the majority of oospores are mature. The duration of this phase differs from year to year. The date of optimum oospore maturation (DOM) was reached on about 24 March 1985, 2 May 1986, and 24 March 1988. Oospores germinated in about 7 days under optimal conditions (20 C, saturated humidity) on these dates. The percentage of oospore germination was roughly comparable in these years (25% in 1985 and 1988, 30% in 1986).

In the third or postmaturation or germination phase, mature oospores form macrosporocysts if climatic conditions are favorable (i.e., temperature above 11 C and free water). During this phase, the proportion of germinated oospores decreased because some oospores had already germinated in the plaster tubes.

Relations among climate, oospore maturation, and downy mildew intensity in spring. A comparison of monthly average temperatures from September through May for the 3 yr (1984–1985, 1985–1986, and 1987–1988) did not reveal important differences except in January (Fig. 2). However, rainfall varied greatly from year to year, especially in fall (September through November) (Fig. 3). Conditions were dry during those months in 1985 (88 mm) and very rainy in 1984 and 1987 (447 and 350 mm, respectively). From December to March, rainfall was similar except in January, which was very dry in 1985 (51 mm) and very rainy in 1986 and 1988 (192 and 232 mm, respectively).

As illustrated by Figs. 1 and 3, rainy conditions during the first months following oospore formation seemed to accelerate the maturation process (1985 and 1988), and dry conditions seemed to delay it (1986).

DOM was not directly correlated with the date of first disease occurrence in spring. DOM was latest in 1986, but primary infections appeared at about the same time in 1985 and 1986 (22 May and 27 May, respectively). This is not surprising, because primary infections depend mainly on conditions after oospore maturation. Nevertheless, disease was more intense in 1985 and 1988 (intensity 4) than in 1986 (intensity 1) despite comparable rainfall and temperatures between the DOMs and the primary infections (Figs. 2 and 3). Consequently, disease level in spring could be correlated with oospore maturity, which depends on autumnal rainfall. Using this hypothesis, we developed a new climate-based model, called "Prediction of Oospore Maturity" (POM), to predict the DOM and consequently disease severity in spring. We then compared simulations of the model from 1977 to 1988 with recorded observations of disease in vineyards.

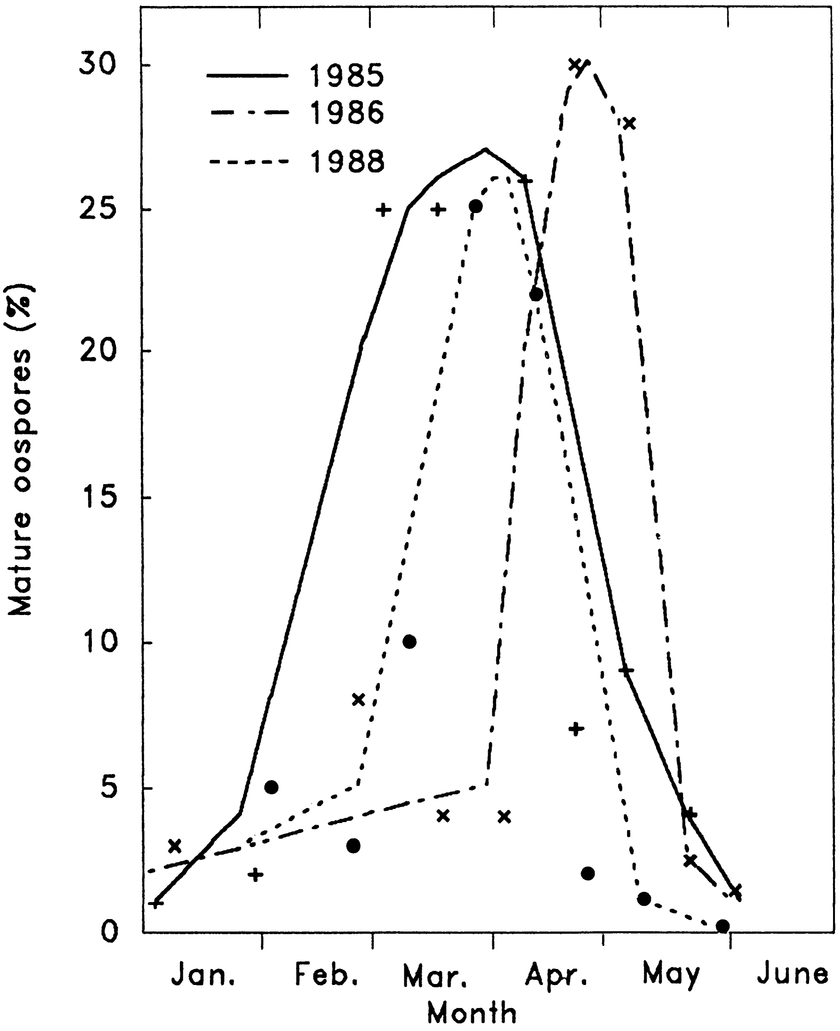

Fig. 1. Dynamics of the percentage of oospore germination at 20 C in 1985, 1986, and 1988 in Bordeaux, France.

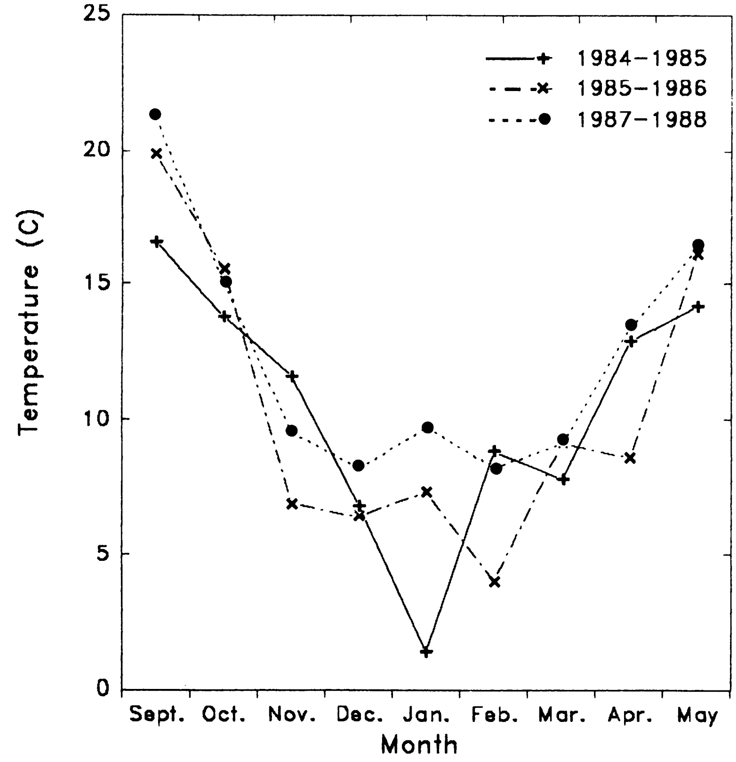

Fig. 2. Average monthly temperatures during oospore maturation in 1984–1985, 1985–1986, and 1987–1988 in Bordeaux, France.

Model development. *Calculation of DOM.* The dynamics of oospore maturation can be represented by a bell-shaped curve (Fig. 1). DOM is the date when the percentage of mature oospores is maximal. Our hypothesis states that this date depends on rainfall during maturation. This relationship can be expressed as $T = A(Im) + B$, where T is the number of days from 1 January to DOM, Im is a maturation index representing the effect of rainfall on maturation, and A and B are coefficients calculated from the results of our study of oospore maturation in 1985, 1986, and 1988.

To calculate Im, we assumed that the amount and distribution (number of rainy days in a month) of rainfall affect oospore maturation and that the effect of rainfall is positive (POS) only within a certain range and is negative (NEG) outside this range. A negative effect could be defined in several ways. We could assume that only a lack of rainfall is negative for oospore maturation. With this definition, any rainfall above a certain threshold (Hm) could have the same positive effect, independent of time. For example, 30 days with 2 mm of rain per day would be similar to 1 day with 60 mm of rain. We rejected this definition in favor of one that takes into account rainfall distribution, and we determined a maximum amount of rainfall (HM) associated with a positive effect. In this second definition, both an excess (EXC) and a lack (LAC) of rainfall are negative for oospore maturation. We hypothesized that $\text{NEG} = |\text{EXC} - \text{LAC}|$ because we reasoned that the development of a "parched" oospore would be favored rather than disadvantaged by an excess of water and vice versa.

We further assumed that the fungus is adapted to local climatic conditions; that is, the quantity of rainfall and the minimum number of rainy days necessary for optimal oospore maturation probably correspond to averages calculated over a long period (e.g., 30 yr or more) in the local area.

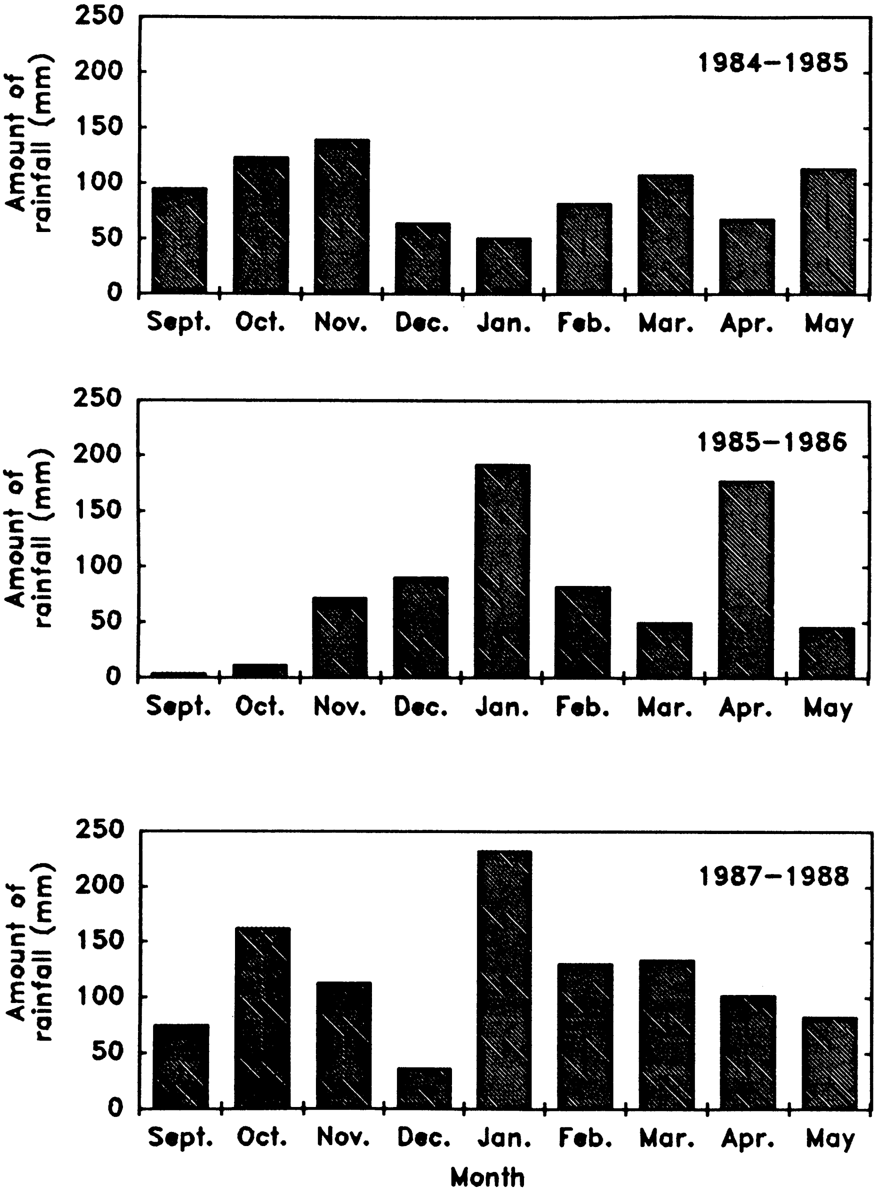

Fig. 3. Rainfall during oospore maturation in 1984–1985, 1985–1986, and 1987–1988 in Bordeaux, France.

To calculate the effect of rainfall on oospore maturation, we used values for the daily rainfall in millimeters (Rd); the monthly average rainfall in millimeters for a 42-yr period (1946–1987) in Bordeaux (RM); the threshold for daily rainfall (Hm), below which lack of rain could exert a negative effect on oospore maturation ($Hm = RM$ divided by the monthly average number of rainy days), calculated over the 42 yr; and the maximum daily rainfall (HM), above which more rainfall could exert a negative effect on oospore maturation ($HM = RM +$ the standard deviation of RM divided by the number of rainy days in the month), calculated over the 42 yr. Thus, for day d, if $Hm < Rd \leq HM$, then $\text{POS}(d) = Rd$; if $Rd < Hm$, then $\text{LAC}(d) = Hm - Rd$; and if $Rd > HM$, then $\text{EXC}(d) = Rd - HM$ and $\text{POS}(d) = HM$.

For a month (M) with n days, $\text{POS}(M) = \Sigma\text{POS}(d)$; $\text{NEG}(M) = |\text{EXC}(M) - \text{LAC}(M)| = |\Sigma\text{EXC}(d) - \Sigma\text{LAC}(d)|$; and $Im(M) = [\text{POS}(M) - \text{NEG}(M)] + Im(M - 1)$, where $Im(M)$ is the maturation index calculated for month M and $Im(M - 1)$ is the maturation index calculated for the previous month. A high value of the index indicates early maturation and therefore a high disease risk in spring.

Im was calculated at the end of each month from September (period of oospore formation) through March (early oospore maturity) for 12 yr (1977–1988). Table 1 compares calculated Im with the observed disease severity (1–4 scale) assessed at the time of bloom in the vineyard. Observed disease intensity and Im were the highest in 1977, 1985, and 1988. By contrast, in 1979, 1981, 1984, 1986, and 1987, Im was low or negative and disease at bloom was weak.

Simple regressions between observed severity and Im for each month (Table 2) show that disease severity increased significantly [$t(\text{calculated}) > t(P = 0.05)$] when Im increased. In addition, this correlation was the highest (correlation coefficient 0.90) for Im calculated at the end of January (IJ). IJ was therefore the most suitable index, suggesting that disease severity for the spring months had been determined much earlier. IJ was thus used to calculate DOM according to the formula stated previously; that is, DOM was deduced directly from IJ, or $T = A(IJ) + B$.

The coefficients A and B were determined from the real values of DOM observed in 1985, 1986, and 1988 and from the IJ values calculated for those years. With 1985 and 1986, we obtained $A = -0.24$ and $B = 117.4$; with 1986 and 1988, we obtained $A = -0.19$ and

$B = 118.4$. We used the means of these values, -0.21 and 117.9, in the model.

Calculations of DOM. Table 3 summarizes our calculations of DOM, using climatic data from the years 1977–1988, and compares our spring disease severity simulations with recorded assessments. The results show that early oospore maturity, incited by a rainy autumn and winter, is correlated with a high risk of downy mildew in spring; similarly, late maturity is associated with a low risk of disease in spring. Thus, DOM appears to be a plausible predictor of risk at bloom.

From the equation of the regression line between observed severity (S) and maturation index IJ ($S = 1.7 + 0.012\,IJ$), we calculated the abscissa of points C1, C2, and C3, which represent the dividing points between the observed severity classes (Fig. 4). The coordinates of these points were C1 (−17,1.5), C2 (67,2.5), and C3 (150,3.5). Then, using the abscissa (IJ) of each point and the equation of the model, we calculated the "limit DOM" for each severity class: 1 May for C1, 14 April for C2, and 27 March for C3.

From these results, predictable severity indices (IG) were defined as follows: If DOM is later than 1 May, then $IG = 1$; only a few unaggressive primary infections would be expected. If DOM is between 14 April and 1 May, then $IG = 2$; scattered primary infections would be expected. If DOM is between 27 March and 14 April, then $IG = 3$; numerous aggressive primary infections are probable. And if DOM is earlier than 27 March, then $IG = 4$; many highly aggressive primary infections would be predicted. Simulated (IG) and observed (S) disease severity were not significantly different (Table 3): χ^2(calculated) = 1.66, less than $\chi^2(0.05) = 21.03$.

DISCUSSION

Our 3-yr study of the dynamics of oospore maturation showed that oospore maturity time (DOM) can fluctuate from year to year. A 1-mo delay was observed in the Bordeaux area between 1985 (24 March) and 1986 (2 May). The assessments of downy mildew severity in spring led us to hypothesize a relationship between DOM and disease severity. To verify this relationship, we needed to study the dynamics of oospore maturation over several years, a long and arduous task. For these reasons, it was desirable to develop a climate-based model to predict DOM.

The POM model runs exclusively with rainfall data recorded after 21 September. It is based on the calculation of a maturation index that evaluates the effect of daily rainfall on oospore maturation. To calculate Im, we considered the hypothesis Strizyk (11) used to elaborate his EPI model, which has been validated in the Bordeaux area over a period of years (5) and also in Italy (7). We hypothesized that rainfall exerts a positive effect on oospore maturation only between certain limits. Below and above these limits, the effect of rain can be negative. Limits were defined using monthly averages of rainfall calculated over a 42-yr period in Bordeaux because, in the EPI model, these data represent the minimum conditions allowing optimal development of the pathogen.

The best results were obtained with Im calculated at the end of January. Moreover, rainfall more favorable to maturation was distributed mainly in fall and early winter, even when oospore maturity was late (1986). Several authors (1–4,10) have noted the role of rain in oospore development, but only Zachos (13) emphasized that early rainfall, during December, could accelerate the end of oospore dormancy. In the POM model, the effect of rain starts as soon as

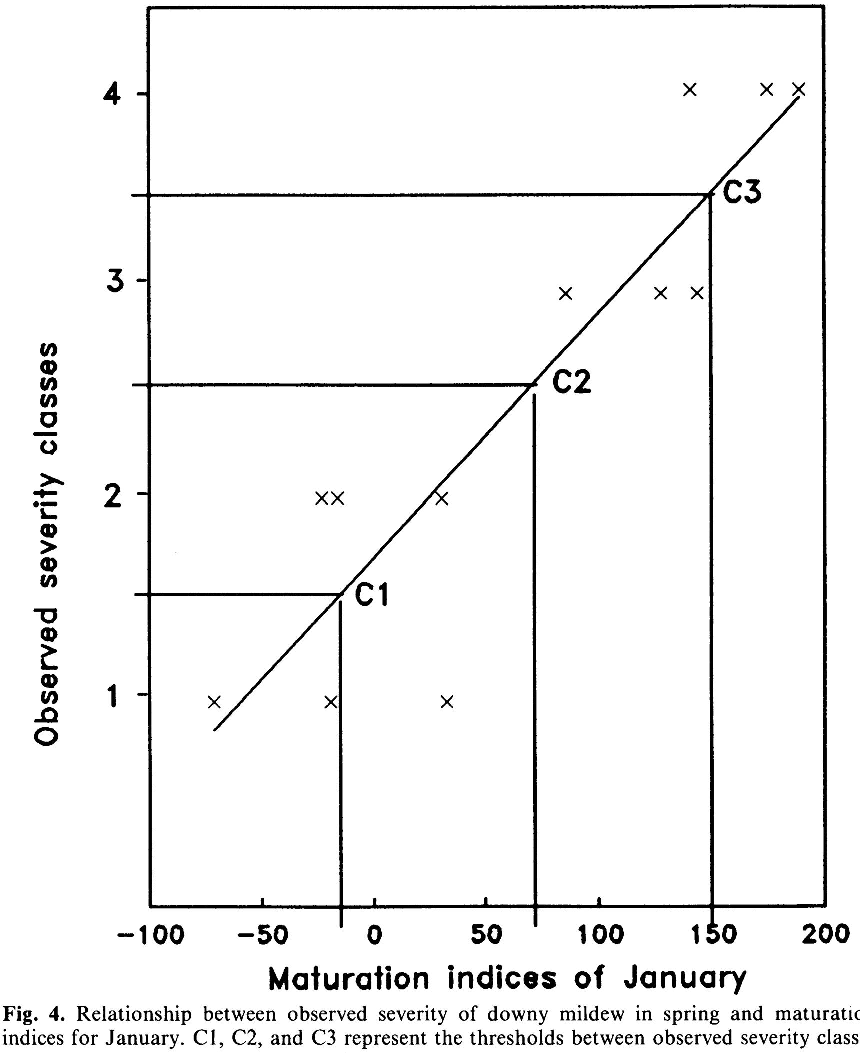

Fig. 4. Relationship between observed severity of downy mildew in spring and maturation indices for January. C1, C2, and C3 represent the thresholds between observed severity classes 1 and 2, 2 and 3, and 3 and 4, respectively.

Table 1. Monthly maturation index (Im), calculated using the "Prediction of Oospore Maturity" model, and observed disease severity at bloom

Im	1977	1978	1979	1980	1981	1982	1983	1984	1985	1986	1987	1988
September (IS)	−4	−24	−12	−9	−24	31	37	−26	32	−26	−31	−3
October (IO)	84	−37	−89	−15	53	97	88	−75	90	−96	−29	113
November (IN)	133	−53	−174	6	2	23	114	−126	184	−98	−61	161
December (ID)	152	−60	−111	76	−24	73	213	−79	168	−93	−24	102
January (IJ)	175	31	−23	128	−16	86	144	33	141	−19	−71	189
February (IF)	258	41	43	79	−42	71	178	68	165	16	−87	270
March (IM)	266	104	96	118	−15	110	191	95	232	11	−66	337
Observed severity	4	2	2	3	2	3	3	1	4	1	1	4

Table 2. Statistical analysis of the correlation between spring disease severity and maturation indices

Month	Regression coefficient	Student's t	Correlation coefficient[a]
September	0.032	3.053	0.69
October	0.012	4.700	0.82
November	0.009	5.829	0.88
December	0.009	4.896	0.84
January	0.012	6.678	0.90
February	0.009	4.850	0.84
March	0.009	5.500	0.86

[a] Significant at 2.228 ($P = 0.05$) and at 4.587 ($P = 0.01$).

Table 3. Spring disease severity calculated using the date of oospore maturity (DOM) and actually observed in vineyards

DOM	Severity	
	Observed	Calculated
22 March 1977	4	4
21 April 1978	2	2
3 May 1979	2	1
1 April 1980	3	3
1 May 1981	2	2
9 April 1982	3	3
29 March 1983	3	3
21 April 1984	1	2
29 March 1985	4	3
2 May 1986	1	1
13 May 1987	1	1
19 March 1988	4	4

oospores are formed.

The most interesting result of this study is the possibility of predicting disease severity in spring using the DOM. Four severity classes were established. In Bordeaux, the risk of severe disease was highest when oospores matured before 27 March. Risk was very low when maturity was reached after 1 May. These observations suggest that disease aggressiveness in spring depends on rainfall distribution from oospore formation (in September) through January.

Although the relation between DOM and disease in spring was observed over several years (1977–1988), the POM model needs further confirmation. It will also be of interest to use DOM to predict the date of primary infections in order to improve forecasts of risk at the time of bloom and to permit better timing of the first fungicide spray.

LITERATURE CITED

1. Capus, J. 1919. Y-a-t'il des périodes critiques pour la contamination du mildiou? Rev. Vitic. 50:330-332.
2. Capus, J. 1919. Recherches sur les invasions du mildiou de la vigne en 1915. Ann. Epiphyt. 4:162-217.
3. Hawker, L. E. 1950. Physiology of Fungi, pp. 209-233. University of London Press, London.
4. Laviola, C., Burruano, S., and Di Graziano, M. 1988. Considerations of the germination of oospores of *Plasmopara viticola* (Berk. et Curt.) Berl. et De Toni in relation to primary infections. Page 81 in: Plant Protection Problems and Prospects of Integrated Control in Viticulture. International Symposium, Lisbon, Portugal, 6–9 June 1988. Centro Nacional de Protecção da Produção Agricola, Lisbon.
5. Maurin, G. 1988. Mildiou: Le modèle est au point. Viti 127:20-22.
6. Molot, B. 1986. La modélisation du mildiou de la vigne: Vers une approche d'une lutte raisonnée? Prog. Agric. Vitic. 103:375-377.
7. Nalli, R., Imbroglini, G., and Leandri, A. 1988. S.P.I. Downy mildew biomathematical model testing on a vineyard in Latium (Italy). Page 85 in: Plant Protection Problems and Prospects of Integrated Control in Viticulture. International Symposium, Lisbon, Portugal, 6–9 June 1988. Centro Nacional de Protecção da Produção Agricola, Lisbon.
8. Ronzon, C. 1987. Modélisation du comportement épidémique du mildiou de la vigne. Etude du rôle de la phase séxuée de *Plasmopara viticola*. Thesis, University of Bordeaux, France. 118 pp.
9. Ronzon-Tran Manh Sung, C., and Clerjeau, M. 1988. Techniques for formation, maturation, and germination of *Plasmopara viticola* oospores under controlled conditions. Plant Dis. 72:938-941.
10. Roussel, C. 1971. Etude du mildiou. Phytoma May:19-24.
11. Strizyk, S. 1983. Mildiou de la vigne: Les données du modèle EPI. Phytoma 350:14-15.
12. Strizyk, S. 1983. La gestion des modèles EPI. Phytoma 353:13-19.
13. Zachos, J. C. 1959. Recherches sur la biologie et l'épidémiologie du mildiou de la vigne en Grèce. Bases de prévisions et d'avertissement. Ann. Inst. Phytopathol. Benaki 2(4):197-355.